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2 IN THE UNITED STATES PATENT AND TRADEMARK OFFICE
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4 In Re: Application of) Art Unit:
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7 Serial No.:)
8 Filed:)
9 For: Brainstem and Limbic)
10 Disorder (BALD))
11 _____)

12 1. Field of the invention.

13 This invention relates to neuro-toxicity and disorders of the
14 central nervous system .

15 Specifically disorders of the brain stem and limbic system of
16 the brain are localized in diagnosis then a specific treatment
17 protocol is outlined.

18 The acronym BALD is used to refer to the brainstem and limbic
19 system disorder in this specification.

20 2. Background

21 The central nervous system is defined as the brain and spinal
22 cord. Associated with the brain are twelve Cranial Nerves which
23 enervate specific areas of the body. These cranial nerves are
24 associated with the brain stem. The brain stem is the area of
25 the brain where the spinal cord connects with the brain itself. The
26 limbic system of the brain refers to a ring of structures that form
27 a border around the brainstem and corpus callosum of the brain.

28 There are a number of recognized disorders of the central

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1 nervous system. These disorders include seizure disorders such as
2 epilepsy, vascular disorders producing headache, and degenerative
3 disorders. CNS degenerative disorders include inherited or
4 degenerative diseases which includes Amyotrophic Lateral Sclerosis
5 (ALS) and nutritional degenerative disease such as vitamin
6 deficiencies and alcoholic abuse. Extrapyrarnidal syndromes are
7 disorders which arise from lesions principally in the basal
8 ganglia. Parkinsonism is another such degenerative disease which,
9 like extrapyramidal syndrome, involves movement disorders. There
10 are also autoimmune disorders such as Myasthenia Gravis which
11 affect the neuromuscular junction, and other immune disorders
12 which affect the Myelin insulation of the nerves, or their ability
13 to produce critical brain chemicals.

14 It is well known that the brain is vulnerable to injuries. It
15 is less well known that the deep structures of the brain are more
16 vulnerable to traction than crush or torsional injuries. Further,
17 the Nervous System is vulnerable to more than just trauma and acute
18 infection. The brain demonstrates vulnerability to changes in
19 levels of glucose, ammonia and other simple molecules. It
20 demonstrates exquisite responsitivity to deprivation of Oxygen and
21 glucose. The brain reacts dramatically to solvents used in
22 mechanical repair areas, to carbon monoxide fumes such as from
23 propane combustion and to toxins such as phenol and toluene. In
24 addition, it demonstrates immune reactivity as in the disease
25 cerebral lupus, to chronic ischemic vascular disease as in as in
26 diabetes, to autoimmune illness as in certain types of cancers, and
27 to oxidative stress from liver failure and concomitant excess of
28 ammonia.

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1 The result in the brain of these combinations of causative
2 conditions, with a final single or ensemble of sub clinical
3 insults is, both central and peripheral neuropathy as well as
4 eventual muscle weakness and limb numbness and pain. Eventually the
5 individual develops a set of nervous manifestations characterized
6 by headaches, fatigue, sleep difficulties, heart rate instability,
7 and gastrointestinal disturbance.

8 The victim of the BALD syndrome described herein develops
9 symptoms other than at the brain and immune system, such as rashes
10 from immune problems and infections, chronic multiple fungal
11 infections, pulmonary, lung and peripheral vascular disease, as
12 well as premature aging on a cellular level. There can be
13 increasing autonomic instability, with body temperature
14 instability, abnormalities of blood flow to organs and skin, and
15 abnormal salivation and sweating . The diagnostic and treatment
16 protocols will examine all these symptoms.

17 All types of diseases, including the neurological diseases are
18 more common in the urban environment, where there is concomitant
19 exposure to industrial and environmental toxic, and polluting
20 substances. People are more frequently developing extreme
21 vulnerability to everyday fumes and substances that many other
22 people tolerate. This is being named Multiple Chemical Sensitivity.
23 There appears to be an increased risk for chronic diseases,
24 including Diabetes, Asthma, and also the Neurological/Neurotoxic
25 diseases. It is known that among the black population, babies who
26 were conceived and had embryonic fetal life in urban America are at
27 more than twice the risk to be stillborn. This is of source
28 neurological death in utero. It is clearly related to the multiple

1 sources for neurotoxicity in our urban environment.

2 A core concept of this invention is that there can be chronic
3 inflammation in neuronal tissue, and loss of adequate Oxidation/
4 energy from oxidative metabolism in critical neural control centers
5 in the brain. Further, this invention seeks to demonstrate that
6 BALD arises from inflammatory immune abnormalities, degradation of
7 connecting axons in the brain, chronic infection especially by
8 fungi and viruses with release of sequestered neuro toxins. This
9 leads eventually to the development of autoantibodies, and chronic
10 electrical instability in neural circuits. Prebirth children would
11 be of course at greatest risk for this type of pathological
12 process. Crib death has just been shown to be the result of fungi
13 acting on the fire retardant in the crib's bedding, causing the
14 production of three types of poison gas. This causes asphyxia in
15 small babies, whose nervous systems are not mature enough to fight
16 off the toxins which are in the environment.

17 In the brain, because of its complexity the neurological and
18 neuroimmune mechanisms, a comprehensive yet focused approach to the
19 core disease mechanisms is necessary. Further the potential for a
20 very insidious onset of signs and symptoms due to the slow
21 accumulation of damage makes sophisticated testing and pathological
22 analysis essential.

23 However, as highlighted by this invention some of the basic
24 pathological mechanisms are now understood, and the treatment
25 approach follows naturally from the pathology. One central
26 mechanism is the condition wherein Oxygen does not sufficiently
27 diffuse from the arterial blood to neurons which need Oxygen to
28 transform Carbon Monoxide which is a neuro transmitter into more

harmless Carbon Dioxide.

Another of the most central of these mechanisms is called excitotoxicity, which is due to breakdown of control over flow of Calcium ions into neurons which leads to neuronal death. This tendency toward excitotoxicity makes the brain and spinal cord vulnerable to process such as reactivation of intra neuronal neurotropic viruses which leads to intracellular damage through mechanisms which are at present unspecified, but much inflammatory damage in the neuron is mediated by Calcium. It is clear that the excessive influx of Calcium into the neuron up regulates a host of secondary messengers that create a number of inflammatory problems. (Baumzweiger, 1998)

It was first noticed that Calcium had a special role in the transmission of information in 1977. (Baumzweiger, 1999) This was first noticed in ALS, where antibodies against L-Calcium channels were found. (Baumzweiger, 1999) This process has been called excitotoxicity. However, there is a second set of problems that arises with this type of damage called oxidative stress, from a combination of diminished availability of Oxygen, reduced ability to use Oxygen to make critical energy storage and biochemical molecules that repair tissue such as methionine which participate in defending healthy tissue. "Free Radicals", which are abnormally electrically charged molecules are one of the results. The liver usually shows the stress from dealing with these processes.

This type of metabolic and neuroimmune stress causes a number of problems to the central nervous system, the liver, kidneys, and to the immune system itself. For the nervous system oxidative stress appears to cause damage to the acetylcholine receptors,

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1 especially the nicotinic receptors associated with motivation,
2 concentration and mental control. (Baumzweiger National Academy of
3 Science Presentation to the institute of Medicine, October 16,
4 1999)

5 The combination of these two problems of toxicity and
6 infectious disease, which play off against each other makes many
7 processes in the nervous system able to reactivate sequestered live
8 virus, fungus and bacteria. When these organisms are captured and
9 sequestered by healthy brain cells, they are generally stopped from
10 reproducing and re-infecting neighboring cells. When they break
11 out, which occurs in multiple sclerosis, there is reactivation of
12 these microbes with inflammatory tissue destruction.

13 Through advanced Lymphocyte testing, viral antibody testing,
14 fungal antibody testing, venous partial pressure of oxygen tests,
15 and testing for autoimmunity, there appears to be a definable
16 condition where the nervous and immune systems meet in the brain
17 stem and limbic system of the brain (BALD). Further there is a
18 tendency towards BALD neurodysimmunity which is a unique mixture of
19 neural dysfunction, immune suppression and multi system
20 autoimmunity which is not seen in any other disease entity.

21 SUMMARY OF THE INVENTION

22 There has been a long felt need for diagnosis and treatment of
23 the above described problems of neuro toxicity. Because the
24 problems involved in localizing pathology to separate areas of the
25 central nervous system the signs and symptoms of the present
26 invention are subtle and often have been dismissed or overlooked by
27 the physician. Thus, one principal object of the present invention
28 is to delineate the clinical signs and symptoms and laboratory

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1 findings resulting from priortrauma or toxic exposure then
2 worsened by specific toxic substances, trauma, or infectious as
3 well as autoimmune disease. Understanding this two pronged
4 pathogenesis will result in changes in medical practice, with a
5 focus on the pathologies due to toxic substances in the modern
6 environment.

7 It is little recognized that the nervous system can and does
8 react with repeated combinations of toxic exposure and infectuous
9 disease, with the onset of tissue damage, even when the individual
10 insults are sub threshold. A protocol for the diagnosis and
11 treatment of the resulting clinical syndrome with reduction of
12 damage to specific areas of the deep brain systems is desirable.
13 With this invention, it will be possible to assess and treat the
14 damage and the vulnerability to further damage from such
15 combinations, as well as their complications such as reactivation
16 of neurotropic virus, and subsequent excitotoxic damage.
17 (Baumzweiger, 1999) A similar tendency toward complexity can be
18 seen in the newly elucidated genetic etiology of schizophrenia and
19 the participation Chlamadia appears to play in heart disease, and
20 which Helicobacter plays in the patho-genesis of peptic ulcers.

21 An extreme case of central nervous system dysfunction caused
22 by toxic substance would be victims of so called nerve gas used in
23 actual wartime environments. It is a principal object of the
24 present invention to localize the central nervous system disorder
25 that is caused by such toxic substances and to describe a treatment
26 protocol for the disorder.

27 Up until the advent of the present invention, it has not been
28 possible to clinically localize BALD central nervous system

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1 disorders to where they originate, that is to specific areas of the
2 brain stem, the basal ganglia connected to the brainstem, the
3 thalamus which coordinates the rest of the brain by referencing
4 these deep structures, and the axonal transmission pathways to the
5 rest of the brain from the brainstem, basal ganglia, and limbic
6 system. Localization of CNS disorders to one or more of these
7 structures is another principal object of the present invention.

8 Many disorders related to the brainstem and limbic system have
9 been mis-diagnosed in the past. A few, such as Parkinsons Disease
10 and Acute Brainstem Encephalitis are well known, but the brainstem
11 and limbic system area has not leant itself to classic techniques
12 of neurological localization. It has had less attention than it
13 deserves. Chronic damage to and inflammation of these areas of the
14 CNS are still not accepted by many physicians, although they are
15 known and accepted by specialists in this area. Further, damage to
16 the brainstem and limbic system is hard to discern, and a very
17 careful, systematic, anatomically based and highly technical
18 approach is needed. It is the principal objective of the present
19 invention to correct this situation not only with a diagnostic
20 protocol but with a treatment protocol as well.

21 The primary purpose of the present invention is to localize
22 and discern the underlying mechanisms which are driving the chronic
23 signs and symptoms, and then treat the resulting disorders. As with
24 all illnesses, eliciting the relevant elements of medical history
25 is essential. As with all illnesses, a history of infections which
26 cause neurological symptoms, a history of nervous system trauma, a
27 history of exposure to environmental, industrial, or wartime toxic
28 or poisonous substances is also essential. This invention involves

1 a diagnostic protocol involving the patient history then a clinical
2 examination to localize the area of nerve tissue damage to the
3 brain stem and limbic systems.

4 Corroborative laboratory tests and other tests can be run` as
5 well.

6 The damage which charged particles can inflict on tissues is
7 typified by the Calcium ion entering neurons and other cells.
8 Calcium ion is the most important signaling ion in the nervous
9 system. With an excess of Calcium in the neurons too little
10 learning occurs or proper response to the environment does not take
11 place, and the subject becomes somnolent. It is one object of this
12 invention to provide a treatment protocol for such a condition
13 which is aimed at reducing Calcium entry into the neuron,
14 decreasing the excess excitability in neural circuits, reducing the
15 activity of intracellular virus and fungus infections, as well as
16 mdication to deal with intencurrent inflammatory infections such as
17 Mycoplasma Fermentas Incognitas, Herpesvirus 2 and 6,
18 Cytomegalovirus, and fungal infections.

19 The present method of diagnosis and treatment of the BALD
20 syndrome is aimed at reduction of brainstem and limbic system
21 dysfunction in two critical ways. First it would reduce the damage
22 from Calcium ion in the nervous system no matter what the cause.
23 Secondly it would aim to repair the damage caused by prior
24 excitotoxic damage to neurons, free radical damage to proteins, as
25 well as inflammatory and infectious damage to critical bodily
26 tissues.

27 DETAILED DESCRIPTION OF THE INVENTION

28 This invention consists of 1) a diagnosis protocol then 2) a

1 treatment protocol for the BALD syndrome.

2 The diagnostic protocol consists of 1) patient history; 2)
3 clinical signs and symptoms and 3) corroborative tests and
4 procedures.

5 Patient history:

6 The key is obtaining a history of exposure or possible
7 exposure to environmental or industrial toxins or poisonous
8 substances. Often there is the history of itching or burning of the
9 scalp, shoulders or neck with possible neck and shoulder weakness
10 which localizes dysfunction to the area of cervical segments C1
11 through C4 inclusive. There is often a history of numbness,
12 weakness, or discomfort from peripheral neuropathy.

13 Signs of the CNS aspects of the BALD disorder are photo phobia
14 and headache. Often there is a history of cognitive deterioration,
15 memory problems, and insomnia. The patient will often experience
16 occasional dizziness on standing, difficulty with walking straight,
17 dizziness, difficulty with swallowing, neck weakness, an odd taste
18 in the tongue usually tinny or metallic. There is usually decreased
19 smell insensitivity for normal smells, but over reaction to very
20 strong smells, and sometimes even olfactory hallucinations. Often
21 there are accompanying gastrointestinal symptoms.

22 Clinical signs and symptoms:

23 Invariably the patient will experience abnormal increase in
24 heart rate on standing, cranial nerve dysfunction, and in advanced
25 cases mild extra pyramidal symptoms. The reflexes in the upper
26 limbs are usually normal which distinguishes the disorder from
27 other CNS disorders such as stroke and peripheral neuropathy. The
28 reflexes and speed present at the knee are brisk, and other limb

1 reflexes such as crossed adduction will be abnormal demonstrating
2 the continued spreading of excess electrical excitability in the
3 nervous system. Further, superficial pathological reflexes such as
4 the glabellar, grasp reflex and finger flexion will appear. The
5 normal infantile reflexes such as the tonic neck, placing reflex
6 and crossed adductor reflex will re-appear. Multiple
7 fasciculations appearing in muscles almost always signifies lower
8 motor neuron dysfunction.

9 Step one in the clinical examination is to examine the sitting
10 to standing heart rate. Usually this is not more than an increase
11 in 10 to 15 beats per minute in the first few seconds after
12 standing.

13 The stethoscope and a stop watch can be used or a pulse
14 oximeter can be employed in this test. One check of the heart
15 rate is made on sitting for 5 minutes, then after standing
16 immediately a check is made at 5 seconds, a check at 15 seconds
17 then a third check at 60 seconds. An abnormal increase on the
18 sitting to standing heart rate indicates dysfunction in the nucleus
19 of Cranial nerve X of the brainstem .

20 The next step in the clinical examination is to examine one or
21 more of the cranial nerves. Dysfunction in a plurality of the
22 cranial nerves is a strong indication that there is damage to the
23 brainstem.

24 Examination of Cranial Nerve I the olfactory nerve:

25 With eyes closed the patient is asked to identify mildly
26 aromatic substances such as vanilla, cologne or cloves. If there
27 is a disorder of Cranial Nerve I this indicates damage to the
28 anterior part of the brainstem.

1 Examination of cranial nerve II the optic nerve:

2 The peripheral vision test is used. The patient is instructed
3 to look straight ahead. Then an object is brought into the
4 peripheral vision of the patient and the patient asked to state
5 when the peripheral object is first seen. Loss of peripheral acuity
6 results from damage to the optic tracts for this retinal area, as
7 they course directly over the inflamed parts of the brain,
8 particularly the cingulate gyrus. In the BALD disorder the upper
9 outer quadrants of the peripheral vision are morer affected than
10 the lower outer quadrants. Paleness of the optic disk or edema of
11 the optic disk is looked for. A patient with inflammation to the
12 brainstem and limbic system, will often experience low tolerance or
13 intolerance to light shown in the eyes. Many cases of photo-
14 phobia are subtle. The patient must be asked very carefully about
15 increased use of dark glasses and hats, or avoidance of the
16 outside. This involves the clinical experience of the practitioner.

17 Examination of cranial nerves III, IV, and VI,, the oculomotor
18 trochlear, and abducens nerves:

19 These three nerves are examined together, since they all act
20 to control the extra ocular muscles. The patient is asked to blink
21 as fast as possible. Fatigue of the levator muscles of the eyelids
22 shows weakness in Cranial nerve III. A penlight is brought
23 from a distance of several feet in front of the patient towards the
24 eyes of the patient to test for visual convergence. The test is
25 positive when there is diplopia up close. There can be observed by
26 hyper convergence with double vision up close along with diplopia.

27 With lengthening of the penlight image distance horizontally,
28 "sparkles" increase in the light of the penlight, or there is

1 sudden darkening of the light or change in light color indicating
2 cranial nerve dysfunction. These signs demonstrate loss of
3 convergence at a distance . The light also is gradually moved
4 backwards from the face, to twenty feet. At a distance a color
5 change in the light can result. The most common color change is
6 because of diffraction effects. At times the light will split into
7 2 lights at a distance. At other times, sparkles will appear or the
8 patient will note the light elongating horizontally. As with other
9 cranial nerves we will describe, there is reduced dynamic range of
10 the reflex control of the cranial nerve responses. This is seen in
11 Cranial Nerves I, III, IV, V, VI, VII, and XII which all carry the
12 special senses.

13 Examination of cranial nerve V, the trigeminal nerve:

14 This nerve carries sensory and motor neurons to specific areas
15 of the face. The patient's facial sensation to pin prick,
16 temperature, and vibration are tested. These are compared with the
17 same sensations on the sternum. To test for damage to the third
18 division of this nerve the three areas of the face and neck
19 enervated by this cranial nerve are tested and compared for
20 sensation,.Sensory loss indicates damage to this nerve. Also small
21 differences in sensation and delayed onset of sensory responses
22 indicate damage to this nerve.

23 Examination of cranial nerve VII, the facial nerve.

24 This nerve is tested for damage by asking the patient to
25 perform various facial movements and checking for loss of sensation
26 inside of the external ear. The pupil of the eye may be wide. There
27 may be fewer wrinkles on one side of the face, and there may be
28 asymmetry to voluntary smiling or forehead wrinkling. Weakness of

1 the nerve can be detected also by asking the patient to detect
2 sweet, salty, and sour substances that are applied to the tongue.
3 Loss of taste sensation definitely would indicate damage to this
4 nerve.

5 Examination of cranial nerve VIII the vestibulocochlear nerve.

6 This nerve transmits sound which allows a person to hear.
7 Using a tuning fork the patient is asked to detect sound of lower
8 frequencies from the tuning fork using air conduction and bone
9 sound conduction. Loss of sound detection indicates damage to
10 this nerve.

11 Examination of cranial nerves IX and X.

12 If cranial nerve IX is damaged the pharynx tonsillar pillars
13 will show diminished sensitivity, especially on the side opposite
14 of the greatest brainstem irritation. Cranial nerve X damage will
15 cause a movement of the soft palate to the side, instead of the
16 normal movement forward, when the tonsillar pillars are stimulated.
17 Normally the soft palate should be symmetrical and should not
18 deviate to either side. When speaking "me, la, ka" from either side
19 of the mouth, there may be a subtle deterioration of pronunciation,
20 particularly on the side of the greatest brainstem irritation.
21 Failure to properly enunciate these phonemes indicates damage to
22 cranial nerve X. Further, these cranial nerves enervate the carotid
23 sinus and the carotid bodies and damage to this nerve can be
24 detected with the carotid body reflex which results in heart rate
25 and blood pressure changes when moving from standing from a sitting
26 position. The orthostatic tachycardia reflex which is reflex
27 tachycardia resulting from change in position from lying to
28 standing and which lasts only a few seconds can be used to detect

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1 damage to cranial nerve X. This reflex tachycardia can be best
2 detected using a pulse oximeter. This cranial nerve controls the
3 gastrointestinal tract, and gastroesophageal reflex so that
4 constipation and diarrhea are very frequent in the BALD syndrome.

5
6 Examination of cranial nerve XI, the accessory nerve.

7 This nerve is a motor nerve enervating the stemocliedomastoid
8 muscle. To test this muscle for strength the patient is asked to
9 turn the head toward one shoulder and to resist attempts of the
10 examiner to move the head in the opposite direction. Then the test
11 is repeated on the other side. Weakness in this muscle indicates
12 damage to this cranial nerve. This nerve which moves the back of
13 the head to the side being tested is the only cranial nerve with
14 ipsilateral cortical connections.

15 Examination of cranial nerve XII, the hypoglossal nerve.

16 This nerve can be tested by asking the patient to push the
17 tongue against either cheek then testing the strength of the tongue
18 by pressing from the outside of the cheek. Fasciculation of the
19 tongue, involuntary movements with the tongue at rest can be seen
20 in advanced disease,

21 Spinal cord Damage

22 The next step in the clinical examination is to test for upper
23 spinal nerve damage. The strength of the trapezius muscle can be
24 tested by asking the patient to push the muscle against the hands
25 of the examiner. This checks for damage to spinal segments C-3 and
26 C-4. Any weakness in the neck muscles especially on flexion of the
27 head indicates weakness of the nerve damage to the brain stem and
28 high cervical cord. Due to viral infection there may be shingles,

1 or loss of sensation on spinal dermatomes, or in some cases
2 weakness below the high cervical spine. There may be decreased
3 strength of respiration, with shallow breathing, and difficulty
4 with ventilation.

5 Motor Examination.

6 The next step In the clinical examination is to check for
7 increased motor tone by moving joints in the arms over their
8 passive range of motion and checking for involuntary muscle
9 resistance. Spasticity or loss of fine motor cc-ordination
10 indicates upper motor neuron damage. Increased motor tone indicates
11 upper motor neuron damage, possibly damage to the brainstem and
12 other brain structures in close proximity to the brainstem and
13 limbic system.

14 Peripheral Nerve Examination:

15 Peripheral nerve damage using the pin prick test and
16 temperature test to check for loss of sensation is useful. A cool
17 tuning fork is handy for checking the limbs, as well as the face
18 for temperature. There is often loss of two point discrimination
19 especially on the side opposite the most affected part of the
20 brainstem. In the advanced stages of the BALD disorder decreased
21 sensation, altered sensation, and delay in experiencing sensation
22 is observed.

23 In the BALD syndrome there is loss of stereognosis, so the
24 patient cannot touch the area of skin the examiner has touched.
25 There is, further, a loss of sensation or dyesthesea, abnormal,
26 uncomfortable sensation on one or both sides of the limbs. There is
27 loss of the ordinary ability to touch one's fingers together behind
28 one's back with the eyes closed in the BALD syndrome. The normal

1 person should be able to do this three times in a row without
2 missing.

3 The next step is to check for abnormal reflexes, such as a
4 mild glabellar reflex. Other superficial reflexes are usually
5 normal.

6 There is usually no Babinski sign. With localized damage to
7 one side of the brainstem and limbic system there may be a partial
8 Babinsky on the opposite side that is damaged.

9 Reappearance of infantile Reflexes:

10 With brainstem damage infantile reflexes appear. The reflexes
11 checked are: crossed extensor reflex, contra lateral reflex arc,
12 deep tendon reflexes, the tonic neck reflex is checked for
13 reappearance, and the infantile grasp reflex is checked for
14 reappearance. The infantile placing reflex is checked for extension
15 of the leg muscles upon rubbing of the shin.

16 The clinical examination must check for lung and bronchial
17 apparatus dysfunction. Prolonged expiration may be heard. The
18 spirometry test is used and a number of these patients with
19 brainstem and limbic system damage show mild restrictive airway
20 disease. Increased auscultation usually points to increased
21 intestinal motility.

22 The next step in the diagnosis protocol is to run laboratory
23 tests to corroborate brain stem and limbic system damage. With
24 neuro toxicity and damage to neurons there will be abnormal levels
25 of evidence of viral and fungal infection. Specific, tests for
26 abnormal levels of virus presence such as the Barr-Epstein virus
27 CMV virus, HHV6 virus, and HHV2 virus can show abnormal
28 vulnerability to viral infections and re-infection. The T4/T8

1 lymphocyte levels can be tested for abnormality in the immune
2 system. On the usual lymphocyte panel tests used to identify
3 immune system dysfunction there will be evidence of both immune
4 suppression as well as evidence of auto immunity in the BALD
5 syndrome. The T(4)/T(8) cell ratio is either too high or too low.
6 The test for NADH is abnormal indicating neurotoxicity and damaged
7 neurons. In a nutshell abnormal results of tests for abnormal
8 antibodies to neuron components reveals damaged neurons.

9 It is possible to run a coagulation panel or serum profile to
10 be used to check for 1) fibrinogen antigen, 2) heparin assay, 3).
11 thrombin/anti-thrombin complexes, 4) soluble fibrin monomer, and
12 5) platelet associated Ig G. Immune system activation of slow cold
13 agglutination and increased free fibrin escaping in to the serum show
14 immune system dysfunction and activation of coagulation,
15 demonstrating a cause for tissue asphyxia or hypoxia. The root
16 cause of this immune system dysfunction is brainstem dysfunction.
17 Further the root cause of abnormal blood clotting is excitability
18 of neurons and blood vessels from excessive intake of Calcium ions
19 into the neuronal cells and blood vessel cells.

20 The next step in the diagnostic protocol is to run a MRI check
21 with a long T-2 sequence, using Gadolinium, on the brainstem and
22 limbic system. Necrotic tissue and gliosis can be observed in this
23 test in severe cases of the BALD syndrome.

24 The next step in the diagnosis protocol is to run a brief
25 neuropsychiatric examination checking for 1) loss of the sense of
26 the familiar, 2) recent onset of obsessive behavior, 3) loss of
27 predictability in behavior, and 4) decreased interpersonal
28 involvement. Emotional disorders may be present such as hysterical

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1 responses to certain events.

2 The treatment protocol consists of ruling in or out of the
3 BALD syndrome then treatment of the core pathology of the syndrome
4 if present. This consists of treatment of the most acute aspects of
5 the BALD syndrome followed by more conservative treatments of the
6 more chronic symptoms or pathology. If diagnosis progresses to
7 brainstem or other neuron damage with significant inflammation the
8 treatment consists of immediate inflammation reduction and
9 stabilization of the immune system. This is followed by
10 conservative treatments as indicated in the diagnosis of the
11 disorder.

12 The treatment proceeds in steps.

13 Step one in the treatment of the disorder would start with
14 drugs that are known to block calcium intake channels of neurons.
15 One such drug that has been used successfully clinically is Nimotop
16 from Bayer Pharmaceutical Company. The chemical name for this drug
17 is nimodipine which has the ability to inhibit movement of calcium
18 ions across the cell membrane. Nimodipine has a greater effect on
19 cerebral arteries than on other arteries, possibly because it is
20 highly lipophilic. This is beneficial for a patient who is
21 normotensive. The usual dosage can be 30 mg. three times daily
22 more or less in the clinical judgment of the physician.

23 Another such drug is Plendil from Astra Merk Laboratories. The
24 chemical name for this drug is feldipine which is known to also be
25 a calcium intake channel blocking agent This is better for a
26 patient who is hypertensive.

27 Step two in treatment of a severe disorder is to proceed with
28 a drug that aids in inhibition of neural activity in the brainstem.

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1 The major inhibitory neurotransmitter in the central nervous system
2 is gamma aminobutyric acid (GABA). Such a drug would be gabatrill
3 from Abbot Laboratories with a chemical name of tiagabine
4 hydrochloride. It is believed that gabatrill blocks GABA uptake into
5 pre synaptic neurons, permitting more GABA to be available for
6 receptor binding on the surfaces of post-synaptic cells. This
7 exerts an anti seizure effect by preventing the propagation of
8 neural impulses that contribute to seizures by a GABA-ergic action.
9 The GABA agonist drug such as gabatrill or tiagabine hydrochloride
10 from Abbot Laboratories should be augmented by another drug
11 Mysoline, an anti epileptic which increases the sensitivity of the
12 GABA receptor neuron complex to GABA.

13 Step three in the treatment of a BALD syndrome involving viral
14 infection to any degree is to administer anti-viral and anti-fungal
15 drugs to the patient. Clinical success has been noted in the
16 administration of Acyclovir which is a generic drug with a chemical
17 name of acycloguanosine. This is a synthetic acyclic purine
18 nucleoside analog. In vitro it has inhibitory activity against a
19 broad spectrum of viruses such as herpes simplex virus types 1 and
20 2, varicella zoster virus, epstein-barr virus and cytomegalovirus.
21 The drug inhibits viral DNA replication.

22 Another drug that has produced good clinical results in the
23 BALD condition is difulcan from Roerig Pharmaceutical Company with
24 a chemical name of fluconazole. This is a synthetic broad spectrum
25 bis-trazole antifungal agent. Still another clinically successful
26 anti-viral drug from Glaxo Wellcome is Valtrex with a chemical name
27 of valacyclovir hydrochloride. Sproonox from Janssen is another
28 drug that can be used successfully as an antifungal agent. This is

1 a synthetic trazole antifungal agent with the chemical name of
2 itraconazole.

3 Step four is to administer antibiotics to the patient to treat
4 for streptococcal or other bacterial infection. Also mycoplasmal
5 infection or parasitic infection should be treated. Antibiotics
6 should be used with great caution keeping in mind that some
7 antibiotics are themselves toxic and can damage already
8 compromised tissues. It must always be understood that the BALD
9 syndrome is a neurotoxic disease and is associated with tissue
10 toxicity in general.

11 Step five is to treat immune disequilibrium and peripheral
12 neuropathy by using Immune Globulin IV from human serum while the
13 patient is still being administered GABA agonists and calcium
14 intake channel blocking agents. This step should be initiated 3 to
15 6 months after ongoing treatment using steps 1 to 5. This is a
16 generic drug which contains 5% immune globulins. The patient
17 should be premedicated with benadryl from Parke Davis
18 Pharmaceutical Company with a chemical name of diphenhydramine
19 hydrochloride. Benadryl reduces or prevents most of the physiologic
20 effects of histamine which includes inhibition of respiratory,
21 vascular and gastro-intestinal smooth muscle constriction,
22 decreased capillary permeability and decreased histamine activated
23 exocrine secretions. Along with the use of immune globulin. IV, the
24 patient should be given the generic drug methylprednisolone which
25 is an adrenal glucococorticoid and which acts as a potent
26 anti-inflammatory agent.

27 Step six in treatment involves administration of
28 anti-coagulants to the patient. Heparin which is a generic drug can

1 leukortine receptor antagonists which act to inhibit
2 bronchoconstriction.

3 To improve oxygenation of the serum Oxygen by face mask or
4 cannula can be administered as well as NADH (ENADA) a generic
5 substance to improve oxygenation. Heparin can also be administered
6 to the patient along with Oxygen. If venous blood tests show
7 partial pressure of Oxygen greater than 30 mm of mercury then
8 Oxygen is not being properly absorbed through the arterial blood
9 system.

10 The brainstem produces carbon monoxide in neuro transmission
11 and this must be combined with serum Oxygen to produce Carbon
12 Dioxide which is a much less harmful substance to neurons and other
13 cells of the body.

14 This is often a very important part of the treatment protocol
15 in the BALD syndrome.

16 Azulfadine should be started for patients with irritable bowel
17 syndrome. This successful drug in clinical usage is from Pharmacia
18 & Upjohn with a chemical name of sulfasalazine. This has an
19 anti-inflammatory property to liver tissue and to tissue of the
20 intestinal walls. This drug is indicated for ulcerative colitis.
21 This should help considerably, especially if it is started at an
22 interval of 3-5 days after the IV anti-microbial medication is
23 given.

24 If there is persistent drooling, motor restlessness and
25 cramping of the muscles, Amantadine Hydrochlyde can be
26 administered.

27 Step nine in treatment involves treatment for pain, depression
28 and psychosis. Numerous drugs work in this area depending on the

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1 symptotology involved. The first choice for analgesia is morphine
2 sulphate a generic dnrg, with good anti-inflammatory properties
3 relative to neurons.

4 Depression can be successfully treated with the generic drug
5 trazedone hydrochloride. This is a monamine oxidase inhibitor which
6 does not stimulate the central nervous system. In the central
7 nervous system it selectively inhibits serotonin uptake by brain
8 synaptosomes.

9 A clinically successful anti-anxiety drug is Klonapin with the
10 chemical name of clonazepam. This drug also acts to prevent
11 seizures.

12 Mag-ox can be administered for magnesium deficiencies and as
13 an anti-acid.

14 All of the steps in the treatment of the BALD syndrome can be
15 monitored by suitable clinical examination and laboratory tests.

16 The above description of the diagnostic protocol and the
17 treatment protocol of the BALD syndrome is for purposes of
18 illustration and not for purposes of limitation. The limitations of
19 the present invention are set forth in the claims

20 What is claimed is:

21
22
23
24 1) A method of diagnosis of a disorder of the central nervous
25 system of the human body that localizes the disorder to the
26 brainstem of the body.

27 2) A method of diagnosis of a disorder of the central nervous
28 system of the human body that localizes the disorder to the